After Bypass Cardiac Surgery, Over 50% of the Patients Developed a Severe Lactic Acidosis While Having Normal Hemodynamics: We Would Like to Put Forward an Hypothesis!

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To the Editor:

With interest, we read the recently published article by Greenwood et al (1) in *Critical Care Medicine* who investigated the anaerobic production of lactate, which was associated with decreased microcirculatory blood flow and decreased mitochondrial respiration following cardiac surgery with cardiopulmonary bypass. In their interesting discussion, they find that over 50% of their patients developed a severe lactic acidosis while having normal hemodynamics, which is similar to cardiovascular surgery patient cohorts in the literature (2). In addition, they find a strong, positive correlation with circulating succinate, citrate, and malate suggesting either an impairment in mitochondrial respiration or an enzymatic deficiency within the tricarboxylic acid (TCA) cycle. Increases in circulating TCA intermediates over time suggest a metabolic bottleneck may occur, which leads to the removal of excess TCA cycle substrates that were unable to be used for oxidative phosphorylation (cataplerosis). While they were arguing about the origin of this phenomenon, we would like to put forward a hypothesis. While during the operation, the patients were sedated with inhaled isoflurane. Unfortunately, we do not know which sedation they received upon ICU arrival, as they were looking at the early postoperative period. In the patients were on propofol in ICU even at normal doses, than they might be an hypothesis. We all know that propofol-induced syndrome is due to high doses of propofol, which is very infrequent (3). What is less known is the fact that low doses of propofol can induce lactic acidosis after cardiac surgery (4). The mechanism by which lactic acidosis occurs has not been elucidated, but in vitro evidence exists to support mitochondrial dysfunction with the poisoning of the electron transport chain and impaired oxidation of long-chain fatty acids as a possible mechanism (5). Indeed, Greenwood et al (1) find a strong, positive correlation with circulating succinate, citrate, and malate suggesting either an impairment in mitochondrial respiration or an enzymatic deficiency within the TCA cycle. The question is obviously by which mechanism? We put forward the hypothesis that if the patients were upon propofol in ICU even at low doses, this might be a potential explanation.

We have still some much to learn about the relationship between propofol and mitochondria, especially after cardiac surgery under bypass (5).

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